Alcohol's Collateral Damage: Childhood Exposure to Problem Drinking and Subsequent Adult Mortality Risk

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Abstract

Alcohol consumption is a well-established risk factor for illness, injury, and death among drinkers. But the consequences of alcohol consumption on individuals other than drinkers, termed collateral damage, are poorly understood. We address this gap by drawing on cumulative inequality theory and examining how exposure to problem drinkers in childhood shapes mortality throughout adulthood. We use data from the 1988-2006 National Health Interview Survey-Linked Mortality Files and estimate Cox proportional hazards models. Childhood exposure to problem drinkers is common (nearly 1 in 5 individuals were exposed) and elevates mortality risk throughout the adult life course. The primary intervening mechanism is risky behaviors, such as smoking, drinking, and reckless driving. Salubrious adult circumstances did not ameliorate the consequences of childhood exposure to problem drinking. The findings—which reveal that the influence of problem drinking is far-reaching and can affect all household members—should inform policies to reduce detrimental effects of problem drinking.

Keywords

alcohol, problem drinking, childhood conditions, mortality, NHIS

Alcohol consumption is a major avoidable risk factor for chronic disease, injury, and death (Rehm et al. 2009). It is the third leading actual cause of death in the United States, after smoking and poor diet and physical inactivity (Mokdad et al. 2004). Problem drinking can affect both drinkers and nondrinkers. The consequences of alcohol consumption for individuals other than drinkers, especially problem drinkers, are presumed to be substantial. While there is a long tradition of research on the immediate or short-term consequences of parental problem drinking on the well-being of children and adolescents, there is comparatively little research on the long-term consequences of childhood exposure to problem drinking when those children become adults. This study draws on cumulative inequality theory (CI theory: Ferraro, Shippee, and Schafer 2009; Ferraro and Shippee 2009) to glean insights into the extent to which exposure to problem drinkers during childhood has enduring consequences for mortality risk throughout adulthood.

BACKGROUND

In the United States and other developed countries, alcohol consumption contributes more to the loss of healthy years of life than does overweight and obesity, physical inactivity, low fruit and vegetable consumption, illicit drug use, or unsafe sex (Ezzati et al. 2004). While the consequences of alcohol consumption on the health of drinkers have been extensively studied, researchers have become increasingly interested in the broader effects on society and on individuals other than drinkers. The societal costs of alcohol consumption (e.g., health care, law enforcement, work productivity) in the United States have recently been estimated to be an impressive 2.7 percent of gross domestic product (Rehm et al. 2009). But some scholars claim that the estimated costs may be conservative because they do not account for many of the

presumed impacts on individuals connected to drinkers (Casswell, You, and Huckle 2011; Connor and Casswell 2012; Giesbrecht, Cukier, and Steeves 2010; Livingston, Wilkinson, and Laslett 2009). The impacts may be widespread and substantial. For instance, the U.S. National Council on Alcoholism and Drug Dependence estimates that more than half of adults have a family history of alcoholism or problem drinking (National Council on Alcoholism and Drug Dependence [NCADD] n.d.). Highlighting the extent of the potential impacts on nondrinkers, a review of studies in New Zealand found that the prevalence of self-reported harm from others' drinking in the past year exceeded the prevalence from own drinking (18 versus 12 percent) (Connor and Casswell 2012).

The impacts of alcohol consumption on the health and well-being of individuals other than the drinker—termed "collateral damage" or "second-hand effects of drinking" (Giesbrecht et al. 2010)—are understudied and poorly understood. Indeed, Livingston and colleagues (2009: page 778) assert that "…with the exception of violence, there is little research into the impact of other people's alcohol consumption on the people around them." Moreover, there is little knowledge about the extent to which exposure to others' drinking has *enduring* consequences for the health and well-being of individuals throughout the adult life course. The focus of extant studies has primarily been on the immediate or short-term consequences among children, adolescents, and more recently on young adults in their 20s (Christoffersen and Soothill 2003). But it is important to understand the *long-term* consequences of exposure to others' drinking. This information is needed to estimate the total social, economic, and health costs of alcohol consumption and to inform public policy and the general public. Interestingly, scholars have underscored the need for this information by drawing parallels with smoking, noting that information on the dangers of second-hand smoke was crucial in the debate and development of

public policy related to smoking (Casswell et al. 2011; Livingston et al. 2009).

The overarching aim of this study is to examine the long-term consequences of exposure to others' alcohol consumption during childhood. Specifically, we use CI theory (Ferraro et al. 2009; Ferraro and Shippee 2009) to reveal the extent to which exposure to problem drinkers during childhood has enduring consequences for mortality risk throughout adulthood, and to identify some of the key mechanisms that explain the association. Consistent with prior research, we focus on exposure to problem drinking, also referred to in the literature as heavy drinking, risky drinking, alcohol misuse, or alcohol abuse.

Assessing Alcohol's Collateral Damage Using Cumulative Inequality Theory

CI theory incorporates the contributions of a broader life course perspective (Elder 1998) and cumulative advantage/disadvantage theory (Dannefer 2003) into a middle-range theory that can guide research on the early-life origins of adult health. "Cumulative inequality theory specifies that social systems generate inequality, which is manifested over the life course via demographic and developmental processes, and that personal trajectories are shaped by the accumulation of risk, available resources, perceived trajectories, and human agency" (Ferraro and Shippee 2009: page 334). We use this theory to examine if and when exposure to problem drinking in childhood produces unequal mortality risk throughout adulthood.

In addition to the overarching framework, several axioms of CI theory motivate our analyses. First, CI theory argues that childhood conditions influence adult outcomes (Axiom 1a), and that family lineage is particularly important because of the shared genetic and living environments (Axiom 1c). Related to this axiom is the notion that early-life exposures can be biologically embedded (Barker 1997; Montez and Hayward 2011). Certain developmental

periods may be “critical” such that exposures during them have an almost certain and irreversible physiological impact. Other periods may be considered “sensitive” such that exposures during them have a likely but potentially malleable impact (Ben-Shlomo and Kuh 2002).

While critical periods are empirically difficult to identify, prenatal exposure to alcohol is one notable exception as it can lead to fetal alcohol spectrum disorders (FASDs). The effects of FASDs are permanent and include a range of conditions including intellectual disabilities, impaired vision and hearing, compromised heart and kidney function, and such behavioral problems as poor impulse control (Centers for Disease Control and Prevention [CDC] n.d.). Problem drinking among family members may also signal shared genetic risks. Indeed, studies suggest that multiple genes influence—but do not determine—alcohol initiation, metabolism, and reinforcing properties (Clark 2006).

Children may also be biologically “scarred” by chronic exposure to others’ problem drinking. Children who live with an alcoholic parent may be at higher mortality risk as adults because of the direct effect of being raised in a risky family environment. Alcoholic adults may provide less parental supervision and may be more likely to neglect or abuse children (Burke, Schmied, and Montrose 2006). These risky environments can create chronic stress, infections, and inflammation, which can in turn result in negative physiological changes (Taylor, Repetti, and Seeman 1997).

Second, CI theory emphasizes the accumulation of inequality over the life course (Axioms 1 and 2). Across the life course, dis/advantages at one point in time increase exposure to subsequent dis/advantages. Because older adults have been exposed to disadvantages (or advantages) for a longer time, differences in mortality risk may be greater among older adults. However, as CI theory notes, the effects of inequality create compositional changes that may

obscure the differences in mortality risk among older individuals. Differences in the effects of childhood exposures on adult mortality across age should therefore be interpreted in light of possible left-censoring.

Third, CI theory contends that inequality need not be confined to one domain (Axiom 2b). Studies among U.S. adults have found compelling evidence that experiences in early life have enduring consequences for adult mortality risk, but this research has either focused on socioeconomic disadvantage (e.g., Pudrovska 2014; Warner and Hayward 2006) or used broad measures of misfortune (Morton, Mustillo, and Ferraro 2014; Schafer, Ferraro, and Mustillo 2011). However, there may be many other household or family characteristics that have long-term effects. One understudied characteristic is problem drinking, which is conceptually distinct from socioeconomic status (SES), family structure, or abuse.

Further, problem drinking may have a range of repercussions on household members that ultimately shape adult health and mortality. For instance, exposure to problem drinking may influence health-related behaviors such as smoking and alcohol use, externalizing behaviors such as aggression, and the development of relationship skills. As CI theory notes, adverse exposures (such as childhood exposure to problem drinkers) can have diffuse effects, and thus this study tests whether problem drinking exposure in childhood increases the risk of death from causes of death that are more commonly associated with risky behaviors, stress, or other origins.

Fourth, CI theory identifies onset, duration, and magnitude of exposure as important influences on life trajectories (Axiom 2c). While we do not have data on the onset of problem drinking exposure, we use reports of duration, quantity, and source of exposure to assess whether adult mortality risks differ across these characteristics. Some evidence suggests that the influence increases with the quantity of problem drinkers in a child's life. To give one example, a study of

children aged 6-15 found that deficits in social competence were greater for children with two rather than one alcoholic parents (Hussong et al. 2005). The study's authors posited that the differential reflects the fact that children with two alcoholic parents lack a potentially compensating effect of a nonalcoholic parent.

Deleterious effects of childhood exposure to problem drinkers may increase with duration of exposure. A recent review concluded that the influence appears to be cumulative, such that the longer children were exposed, the greater the impact on their health and well-being (Burke et al. 2006). Studies that have combined quantity and duration of exposure to problem drinkers into a single index have also found a strong, inverse relationship between exposure and personal well-being and self-reported health in adulthood (Casswell et al. 2011).

It is conceivable that the closer the family relationship, the stronger the mortality effect of exposure to problem drinking in childhood. This idea is related to the life course principle of "linked lives" (Elder 1998). In other words, the stronger the links, the stronger the effects. For example, problem drinking among parents may be more important than problem drinking among more distant relatives, including grandparents, aunts and uncles, and cousins.

Fifth, CI theory rejects a deterministic view of inequality, noting that human agency and resources can alter trajectories over the life course (Axiom 3). This axiom parallels the pathway framework, which theorizes that childhood circumstances operate *indirectly* through other factors—such as SES, health behaviors, and psychosocial resources—that then affect adult health and mortality (Montez and Hayward 2011). For example, being raised by a problem drinker may disrupt adult achievement processes, which can have substantial consequences for health and longevity (Phelan et al. 2004). A review of studies that interviewed children of problem drinkers found that the most prevalent theme was educational failure (Tunnard 2002).

Children stated that they often arrived late to school or were kept home to care for parents, had difficulty concentrating because they were too tired from events at home, had parents who showed little interest in their schoolwork, and often changed schools due to family separation.

Individuals chronically exposed to problem drinkers in childhood may also be disposed to engage in unhealthy behaviors during adulthood. As suggested by social learning theory, they may imitate their parents' drinking behavior (Bandura 1977). Nonetheless, some adults who have been surrounded by problem drinkers during their early lives may instead choose to abstain from drinking (Rogers et al. 2013). Additionally, adolescents may develop unhealthy coping behaviors from family and friends—such as problem drinking, smoking, and overeating—to alleviate stress, dysfunction, hardship, and neglect experienced in the childhood home (Pearlin 1989).

Another indirect pathway may occur through psychosocial resources. Families headed by problem drinkers often experience dysfunction, perceive their home environment to lack cohesion, have few routines and rituals, exhibit high levels of arguing and unresolved conflict, and express less warmth, caring, and positive feelings (Burke et al. 2006). These environments can compromise children's development of psychosocial resources, such as self-esteem, a sense of personal control, and social competence (Hussong et al. 2005) which, in turn, shape health. The environments may also hinder development of salubrious social relationships, such as stable and supportive marriages, vital resources for good health (Umberson and Montez 2010).

Although the family is a major source of social support and is generally protective against mortality, it can also be a staging area for conflict, strain, and bad behavior. Spouses can act as co-conspirators, encouraging the other spouse to indulge in poor diet, inactivity, risky driving, violent behavior, criminal activities, drug use, tobacco consumption, and excessive drinking. In addition, alcohol abuse by one spouse may lead to alcohol abuse by the other spouse. Thus, in

this study we examine whether the protective effect of marriage on mortality is smaller for adults with a spouse or partner who is a problem drinker.

In addition to serving as pathways, experiences may moderate the effect of childhood experiences on adult health. For instance, the health of adults who were exposed to a problem drinker in childhood may be more vulnerable to stressors such as poverty and marital disruption in adulthood than adults who were not exposed. On the other hand, their health may disproportionately benefit from protective buffers in adulthood such as prestigious occupations and stable marriages. Educational attainment may be a particularly central buffering resource for adults exposed to problem drinkers in early life because adults with higher levels of SES tend to use effective coping strategies and have higher levels of self-esteem, sense of mastery, self-efficacy, and problem-solving skills (Aneshensel 1992).

Several studies have found support for moderating effects suggested by CI theory. For example, Barker and associates (2001) reported that having a low income or manual occupation disproportionately elevated men's risk of coronary heart disease if they were thin at birth. Support for a moderating effect also comes from studies finding that positive social, familial, and external supports increase one's resilience to the consequences of others' problem drinking (see review in Burke et al. 2006).

Sixth, CI theory considers the importance of *perceptions* in addition to absolute indicators of advantage or disadvantage (Axiom 4). CI theory notes that individuals evaluate their positions, resources, and trajectories in comparison to others and this evaluation influences future trajectories. In assessing the influence of problem drinking exposure, our measure reflects the individual's perception of problem drinking, rather than an absolute indicator of alcohol consumption. Some individuals may have been sensitive to others' alcohol consumption, while

other individuals may have been unaware of the habits of household members. Through capturing the perspective of the individual, we incorporate the perception of this disadvantage.

Aims and Hypotheses

The study addresses the following six questions:

(1) To what extent is childhood exposure to a problem drinker associated with mortality risk in adulthood? We expect that adults who were exposed to a problem drinker during childhood have a higher mortality risk than individuals who were never exposed during childhood.

(2) Which mechanism(s) explain the association between childhood exposure to a problem drinker and adult mortality? In other words, does the exposure leave a direct biological imprint, or set in motion trajectories of socioeconomic, behavioral, and psychosocial disadvantages, or both? We expect that both types of mechanisms are at play.

(3) Do resources or behaviors in adulthood moderate the association between childhood exposure and adult mortality? We expect that the effects of exposure will be reduced for those with greater resources and healthier behaviors.

(4) To what extent does the association between childhood exposure to a problem drinker and adult mortality change across the life course? Does it strengthen, weaken, or persist with age?

(5) How are the quantity, duration, and source of childhood exposures to problem drinkers related to adult mortality? We expect that exposure to a greater number of problem drinkers, and a longer duration of exposure, will each elevate adult mortality risk. We also hypothesize that exposure to problem drinking by parents elevates mortality more than problem drinking by other relatives.

(6) To what degree does childhood exposure to problem drinking elevate risk for mortality due

to heart disease, cancer, respiratory causes, external causes (e.g., homicide), and other causes? We expect that exposure to problem drinking elevates the risks of death from each of these major causes, and in particular, external causes.

Together, the findings provide new and important insights into the long-term, collateral effects of problem drinking among U.S. adults.

DATA AND METHODS

Data

We use data from the 1988 National Health Interview Survey Alcohol Supplement (NHIS-Alcohol), which was cosponsored by the National Institute on Alcoholism and Alcohol Abuse (Schoenborn 1991). The NHIS-Alcohol includes an extensive set of questions on past and current alcohol use by the respondent and other key individuals such as parents, siblings, spouses, and partners. The NHIS data are collected by the U.S. Census Bureau using a complex multistage sampling frame that provides nationally representative data on the noninstitutionalized U.S. population. Although the 1988 NHIS-Alcohol interviewed 43,809 individuals aged 18 and over, we focus on the 41,308 adults aged 21 and above who met the legal drinking age.

The 1988 NHIS-Alcohol was linked to the National Death Index (NDI) as part of the broader 1986-2006 NHIS Linked Mortality File (NHIS-LMF). Specifically, in 2010, the National Center for Health Statistics linked the NHIS respondents to death certificates in the NDI through December 31, 2006 using a probabilistic matching algorithm (National Center for Health Statistics [NCHS] 2009). Thus, respondents' vital status was monitored from their NHIS survey until death or the end of 2006 for survivors. Over the 1988-2006 period, 10,494 individuals in

our analytic sample died, roughly 25 percent of our sample. The exceptionally rich and detailed information on alcohol use, including retrospective questions, the large nationally representative sample, the wide age range of adults, and the extended mortality follow-up are major strengths of this dataset.

Mortality

The main outcome of interest is death from all causes. To illuminate the mechanisms that link childhood exposure to problem drinkers to subsequent mortality risk in adulthood, we also examine the risk of death from five leading causes. We use the classification of causes of death from the current International Classification of Diseases (ICD)-10 codes (World Health Organization [WHO] 2007) and separately examine the risk of death from heart disease (I00-I13, I20-I151); malignant neoplasms [cancer] (C00-C97); chronic lower respiratory diseases and lung cancer (J40-J47, C33-C34); external causes (V01-X59,Y85-Y86,*U03, X60-X84,Y87.0, *U01-*U02, X85-Y09, Y87.1); and all other causes. To provide additional insight, we examine the following more detailed causes: cancer excluding lung cancer (C00-C32, C43-C97) and subgroups of external causes including transport accidents (V01-V99, Y85), nontransport accidents (W00-X59, Y86), intentional self-harm (suicide; *U03, X60-X84, Y87.0), and assault (homicide; *U01-*U02, X85-Y09, Y87.1). We examine all cancers versus cancers excluding lung to roughly distinguish smoking-related cancers from other cancers. Our codes are based on the major 113 selected causes of death (NCHS 2009).

Childhood Exposure to Problem Drinkers

Our key exposure of interest is living with a problem drinker or alcoholic during the first 18

years of life. After being told that "People have different opinions about heavy, moderate, and light drinking," respondents were asked "When you were growing up, that is, during your first 18 years, did you live with anyone who was a problem drinker or alcoholic?" Because problem drinker and alcoholic were defined by the respondents, the designations may not fit strict clinical definitions (Schoenborn 1991) but may nevertheless be valuable in identifying subsequent health and survival outcomes. Respondent assessments of others' problem drinking are often used (e.g., Burke et al. 2006) and correlate with indicators of respondents' health and well-being (Casswell et al. 2011). We code all respondents who lived with a problem drinker or alcoholic in childhood as 1, and all other respondents as 0.

Respondents stating that they lived with a problem drinker or alcoholic during childhood were then asked to identify their relationship to the individual (e.g., parent, sibling, other relative). This survey allows the respondent to identify up to five individuals. Furthermore, it asked respondents to state how long they lived with each individual while they were a problem drinker or alcoholic. For simplicity, we hereafter refer to these individuals as a "problem drinker." Using this information we create three additional measures of childhood exposure. The measures include the number of problem drinkers the respondent ever lived with when aged 0-18 (0, 1, 2 or more), the relationship to each problem drinker (parent, sibling, other relative), and the number of months the respondent lived with a problem drinker. Because respondents may have lived with more than one problem drinker, we coded the length of time for problem drinker who lived with the respondent the longest.

Hypothesized Mediators

We hypothesized above that three types of adult circumstances—SES, adult health behaviors,

and psychosocial resources—partly mediate the association between childhood exposure to problem drinkers and adult mortality risk. We include two indicators of adult SES, educational attainment and family income. Education and income are continuous measures; the former reflects years of educational attainment and the latter captures the family income in dollars in the past year. For multivariate analyses, we take the log of family income to normalize the distribution.

The two key adult health behaviors are smoking and drinking. We code drinking status at the time of the interview as abstainer, former drinker, lifetime infrequent drinker, and current drinker. Among current drinkers, we code the average volume into less than 1, 1 to less than 2, 2 to less than 3, and 3 or more drinks per day (for similar coding, see Rehm, Greenfield, and Rogers 2001). All statuses are compared to the category of the lightest current drinkers. Smoking is categorized into those who reported never smoking, having previously smoked, and being a current smoker.

The third group of mediators that we examine reflects psychosocial resources. We include a categorical measure of marital status at interview, coded as never married, married, divorced, and widowed. Marital status likely reflects an indirect pathway linking early exposure to problem drinking to adult mortality, based on such characteristics as social skills, emotional intelligence, conflict resolution, and a source of social support**.** We include an indicator of whether the respondent had ever been married to or lived with a problem drinker or alcoholic as an adult (yes=1, no=0), which may capture the quality of the relationship to some degree.[1]

A few respondents were missing data on one or more mediators. We imputed missing data using the "mi" package in Stata, allowing us to retain all individuals in all analysis. We impute 130 values (0.3%) for education, 5,246 values (12.7%) for income, 42 values (0.1%) for

smoking status, 1,051 values (2.5%) for drinking status, 41 values (0.1%) for marital status, and 347 values (0.8%) for having ever been married to a problem drinker values. All independent and dependent variables are used to inform imputation, as are auxiliary variables (income [less than $20,000, greater than or equal to $20,000], and region).

Statistical Analyses

We estimate a series of Cox proportional hazards models. The models estimate the risk of death during the follow-up period from age and the predictor variables. To account for age, the models use age at interview and the duration of exposure to the risk of death, which is the time elapsed from the interview until death or censoring at 2006. We begin with a baseline model that includes the main predictor of interest—a binary indicator of childhood exposure to a problem drinker—along with basic control variables, sex (0=female, 1=male) and race/ethnicity (non-Hispanic white [omitted reference], non-Hispanic black, Hispanic, Non-Hispanic Asian, and other). We then progressively add the three groups of hypothesized mediators (SES, health behaviors, psychosocial resources) to assess the extent to which they attenuate the association between childhood exposure and adult mortality risk. All analyses account for the complex sampling design of the NHIS.

RESULTS

Table 1 shows descriptive statistics of exposure to problem drinking during childhood and adulthood. Nearly one in five adults reported having lived with a problem drinker during their first 18 years of life. It is much more common to have lived with one rather than two or more problem drinkers during childhood. Parents were the most likely problem drinker relative during

a person's first 18 years of life. Respondents had lived with a problem drinker for an average of about 28 months. This number reflects the influence of the approximately 82 percent of the sample that did not report living with a problem drinker and therefore had a value of 0 months; the average is considerably higher among those who reported ever living with a problem drinker (154.4 months or nearly 13 years).

Table 1 about here

Table 1 also presents variations in problem drinking by other covariates. Individuals were likelier to have lived with problem drinkers during their childhood if they were younger rather than older; female rather than male; non-Hispanic white rather than other race/ethnic groups; divorced rather than married, widowed, or single; low-income rather than high-income; current smokers rather than never or former smokers; and former or current heavy drinkers rather than abstainers or infrequent drinkers. Lastly, nearly a third of those who have ever married or lived with a problem drinker had childhood exposure to a problem drinker, and nearly two-fifths of those currently living with a heavy drinker reported this exposure. Although these results are informative, they do not control for other covariates. Next, we turn to multivariate analyses.

Table 2 displays the risk of death associated with exposure to problem drinking in childhood. Compared to respondents who had not lived with a problem drinker during their formative years, respondents who had done so experienced 18 percent higher mortality (hazard ratio [HR]=1.18) over the follow-up period, with controls for sex and race/ethnicity (Model 1). This mortality risk declines modestly to 16 percent with additional controls for SES. The risk further attenuates with controls for respondents' current drinking and smoking status (Model 3). In fact, the ln(HR) for living with a problem drinker during childhood is reduced by 35% with controls for drinking and smoking status ((ln[1.16] - ln[1.10]) / ln[1.16] * 100). Adding

psychosocial measures—including marital status and an indicator of ever married to or lived with problem drinker as an adult—does not change the effect of childhood exposure much, but these factors themselves have significant effects on mortality risk. The full model (Model 4) shows that the risk of death associated with living with a problem drinker during a person's first 18 years of life is elevated by 9 percent, net of adult socioeconomic, behavioral, and psychosocial controls.

Table 2 about here

We also examined whether adult circumstances, such as SES, moderated the association between childhood exposure to a problem drinker and adult mortality risk (results not shown). Models separately interacted adult drinking status, marital status, education, income, sex, and smoking status with childhood exposure, and only one smoking status interaction term was significant. Our results indicate, for example, that the mortality risks associated with childhood exposure to problem drinking are similar for both males and females and for low- and high-educated adults. The one exception was smoking status. Being exposed to problem drinking in childhood had a disproportionate effect on the mortality risk of current smokers.

Childhood exposure to problem drinking is stronger among younger than older individuals (Table 3). Among individuals exposed to problem drinkers in childhood, the risks of death were 36 percent higher for adults aged 21-44, 23 percent higher for adults aged 45-64, and 10 percent higher for adults aged 65 and older. After adjusting for the three groups of adult mediators, the elevated mortality of adults 65 and older was no longer significant. The elevated mortality risks among adults 21-44 and 45-64 were attenuated to a similar magnitude and remained statistically significant (HR=1.14 and HR=1.11, respectively).

Table 3 about here

Table 4 illustrates the influence of different types of exposure to problem drinking in childhood. As in the previous table, modeling begins with a baseline model (which controls for sex and race/ethnicity), then sequentially adds controls for SES (Model 2), health behaviors (Model 3), and psychosocial factors (Model 4). Importantly, most measures of exposure to problem drinking—including the number of problem drinkers, the individual's relationship with them, and the duration of exposure—elevate a person's risk of death. Controlling only for sex and race, and compared to those who did not live with a problem drinker, those who lived with one problem drinker experienced 17 percent higher risk of death, and those who lived with two or more problem drinkers experienced 24 percent higher risk of death over the follow-up period. When examining the relationship to the problem drinker, we found that exposure to parental and others' problem drinking (but not sibling problem drinking) elevated adult mortality risk; and only parental problem drinking remained significant after controlling for the adult mediators (HR=1.09 in Model 4). When examining duration of exposure we found that the more years that a person had lived with a problem drinker in childhood, the greater the mortality risk. For instance, compared to adults who had not lived with a problem drinker, those who had done so for 12 or more years experienced a 12 percent higher risk of death over the follow-up period, net of adult sociodemographic, behavioral, and psychosocial factors.[2]

Table 4 about here

Table 5 displays the association between childhood exposure to problem drinking and specific causes of death. The baseline model (Model 1) shows that individuals who lived with a problem drinker during childhood were more likely to die from all major causes we examined: heart disease, cancer, chronic lower respiratory diseases and lung cancer, and especially external causes. The elevated risks were most pronounced for two types of external causes—transport and

non-transport accidents. Compared to those who never lived with a problem drinker in childhood, those who did experienced a striking 73% higher risk of death from transport accidents (roughly 93% of transport accidents are motor vehicle crashes (Hoyert and Xu 2012)). In comparison, these adults had a 15% higher risk of death from heart disease and from non-lung cancers.

Models 2-4 progressively control for adult socioeconomic conditions, health behaviors, and psychosocial resources. Poorer health behaviors of individuals exposed to problem drinkers in childhood explains a large portion of their elevated risks of death. After controlling for all three groups of adult mediators, the elevated risk remains significant ($p<0.05$) for only one cause of death—transport accidents. Taken together, these findings indicate that exposure to problem drinking in childhood elevates mortality risk in adulthood in large part through risky behaviors, such as smoking, heavy drinking, and reckless driving.

Table 5 about here

DISCUSSION

Drawing on CI theory, this study provides new evidence about the long-term consequences of exposure to problem drinkers in childhood on the risk of death in adulthood. We find that exposure to problem drinkers in childhood is not only fairly common (nearly 1 in 5 U.S. adults report being exposed) it also has enduring and pernicious consequences on the risk of death throughout adulthood. For instance, adults aged 21 and older who were exposed to problem drinkers in childhood had an 18 percent greater risk of death compared with peers who were never exposed. The results should inform the development of strategies and policies to reduce the collateral damage or second-hand effects of problem drinking.

Several findings are particularly noteworthy. First, the main mechanism through which exposure to problem drinkers in childhood elevates mortality risk in adulthood is risky behaviors. Among the three categories of mediating mechanisms we examined—adult socioeconomic resources, health behaviors (smoking, alcohol consumption), and psychosocial resources—health behaviors were the most important. We found additional evidence for a behavioral explanation when replicating the analyses for major causes of death. Specifically, while individuals exposed to problem drinkers in childhood were significantly more likely to die from all major causes we examined, the elevated risks were most pronounced for causes related to smoking and accidents (especially transport accidents), the latter of which may reflect careless and reckless driving. Several factors could explain why adults exposed to problem drinkers in childhood disproportionately engage in risky behaviors. Consistent with social learning theory, they may be imitating the behaviors of their parents (Bandura 1977). Risky behaviors may also be an externalizing response to being raised in harsh, chaotic, or abusive family environments (Felitti et al. 1998; Taylor et al. 1997). Further, these behaviors may signal biological embedding of early environments. For instance, one feature of FASD is poor impulse control (CDC n.d.). Impulsiveness is, in turn, associated with a host of risky behaviors including marijuana use (Simons and Carey 2002), aggressive driving (Dahlen et al. 2005), and higher levels of alcohol use (Patock-Peckham and Morgan-Lopez 2006).

Another important finding is that exposure to problem drinkers in childhood elevates the risk of death throughout early, mid, and even late adulthood; and the elevated risks are inversely associated with age. Five factors could explain the age-graded pattern. One factor is the well-documented decline in risky behaviors with age, which may be particularly relevant here given that these behaviors are the main mechanism through which early exposure to problem drinkers

elevates adult mortality risk. For instance, the prevalence of alcohol abuse and dependence declines with age (Grant et al. 2004), as does thrill-seeking, risk-acceptance, aggressive driving (Turner and McClure 2003), and impulsiveness (Lyvers, Duff, and Hasking 2011). Second, the age patterns may reflect a decline in the social acceptability of risky behaviors among older adults, as well as neurobiological changes with age (Lyvers et al. 2011). A third factor that might contribute to the age-graded pattern is mortality selection. As CI theory warns, selection may alter the composition of study populations which can create the appearance of shrinking inequalities with age. Fourth, period and cohort influences may be at play. For instance, the oldest individuals may have grown up during the Prohibition era (1920-1933), when the meaning and source of "problem drinking" may have differed from more recent times (Blocker 2006). Finally, high ambient mortality at the older ages—with high mortality risk from most chronic conditions—may soften the effects of exposure to problem drinking in childhood.

The study also provides insights into the extent to which the source, quantity, and duration of exposure to problem drinkers in childhood matters. We found that longer exposure to problem drinkers in childhood contribute to increased mortality risk, which suggests that chronic exposure is especially problematic (see also Burke et al. 2006). In addition, we found some evidence that being exposed to two or more problem drinkers elevated mortality risk marginally more than being exposed to one problem drinker. Source of exposure also seems to matter, with exposure to parental and other relative drinking having a stronger effect than sibling drinking, which supports the notion of linked lives (see also Elder 1998).

One surprising finding is that favorable adult circumstances (e.g., high education, high income, marriage) did not ameliorate the deleterious consequences of childhood exposure to problem drinking on adult mortality risk. Attaining higher education and having a higher income

did not moderate the damage caused by childhood exposure to problem drinking. Only smoking moderated the impact of childhood exposure on adult mortality risk, and it did so in a synergistic manner. The elevated mortality risk of adults who were exposed to problem drinking in childhood was even greater for adults who smoked, which is disconcerting because these adults have a high likelihood of smoking. Drinking and smoking status attenuate the increased risk, and adults who had been exposed to problem drinking in childhood were more likely to smoke and drink, but we cannot state definitively that exposure to problem drinking in childhood results in drinking and smoking as an adult. Nevertheless, we can highlight this combination of risky factors: exposure to alcohol abuse, and current smoking and drinking.

This paper addresses several goals of *Healthy People 2020* related to reducing if not eliminating the almost 80,000 deaths per year attributed to alcohol by reducing the proportion of adults who drink to excess or engage in heavy episodic drinking (US Department of Health and Human Services 2014). Our results indicate that the current numbers of deaths attributed to alcohol are underestimated because they do not take into account the indirect effects of problem drinking—including exposure to problem drinkers—on individuals connected to the drinker. It is useful to consider alcohol-related problems across the entire life course. Policymakers should consider ways to reduce the detrimental effects of exposure to problem drinking among children, such as targeting problem drinking among parents or within family environments. For example, interventions that improve family functioning, provide external supports to the family (such as a stable adult figure), and teach parenting skills can soften the impact of parental problem drinking (see review in Burke et al. 2006). These policies are particularly important given our results which suggest that it is difficult to mitigate the elevated mortality risk associated with childhood exposure to problem drinking when those individuals become adults. Reducing exposure or the

effects of exposure could reduce alcohol-related deaths.

Despite the many strengths of NHIS-Alcohol for our analyses, the study has four limitations that merit mention. First, there may be left censoring that we cannot control for. Some children who were adversely affected by problem drinking of others in the family may have died or become institutionalized (in prisons, jails, mental institutions, or drug rehabilitation facilities) and therefore missed by the survey. Second, we do not have time-varying covariates. We have information about whether the respondent had ever been married to or lived with a problem drinker, but we do not know whether the respondent was still living with the problem drinker at the time of the survey, had gotten divorced, or had been widowed. We do not believe this to be a major limitation because it would lend itself to conservative parameter estimates. Third, although our analyses capture many of the main social and behavioral determinants of health and mortality, we lack information on other potentially important mediators, such as mental health and social support networks. We also lack information on the broader childhood environment, such as socioeconomic conditions and family composition. While this information could be valuable, the effects of parental alcohol abuse on children's health and well-being in adulthood seem to persist even when controlling for childhood SES (Christoffersen and Soothill 2003). Finally, the retrospective questions may have been influenced by recall bias. For instance, adults who were already ill might have selectively remembered more exposure to problem drinking.

Conclusion

Alcohol abuse is a major preventable cause of death that can directly affect the problem drinker, and can also indirectly affect other friends and family members, including children. Just as second-hand smoking can have harmful effects on those exposed to the smoke, so too can

exposure to alcohol abuse as a child (e.g., Giesbrecht et al. 2010). A more thorough understanding of the relationship between alcohol consumption and mortality will contribute to better social policies to reduce or prevent alcohol abuse (see Anderson, Chisholm, and Fuhr 2009). Such policies can lengthen the lives, not only of the problem drinkers themselves, but also of their children, spouses, and other family members.

NOTES

1. Although measures of depression or psychological well-being could reflect additional pathways, such measures are not available on the dataset.
2. We categorize number of years lived with problem drinker(s) into 0, 1-11, and 12 or more years to distinguish between those who have never lived with a problem drinker (the 0-year category); address the skewed distribution; and demonstrate a strong, graded, and statistically significant relationship. The continuous measure was also statistically significant, but the effects were small, in part because the variable is skewed, and because the metric is small (that is, a 1-year increase in living with a problem drinker should reasonably contribute to a fairly modest increase in the risk of death).

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TABLE 1. Descriptive Statistics, U.S. Adults Ages 21 and Over, 1988

	Lived with problem drinker during first 18 yrs	Number of problem drinkers lived with			Problem drinker relation			# months lived with problem drinker
		0	1	2+	Parent	Sibling	Other relative	
Population	18.1%	81.9%	15.4%	2.7%	16.2%	1.4%	1.5%	27.9
Died during follow-up	12.7%	87.3%	11.2%	1.4%	10.9%	1.1%	1.2%	21.1
Sociodemographic factors								
Age								
21-44	22.5%	77.5%	18.7%	3.7%	20.3%	1.6%	1.8%	33.2
45-64	16.2%	83.8%	14.2%	2.1%	14.6%	1.1%	1.2%	27.4
65+	8.4%	91.6%	7.7%	0.7%	6.8%	0.9%	1.0%	14.1
Sex								
Male	16.5%	83.5%	14.2%	2.3%	14.6%	1.2%	1.4%	25.1
Female	19.3%	80.7%	16.3%	3.0%	17.4%	1.4%	1.5%	30.1
Race/ethnicity								
Non-Hispanic white	18.6%	81.4%	15.8%	2.9%	16.8%	1.3%	1.3%	28.7
Non-Hispanic black	16.3%	83.7%	14.3%	2.0%	13.5%	1.2%	2.3%	24.7
Hispanic	17.9%	82.1%	15.5%	2.4%	15.7%	1.7%	1.7%	28.9
Non-Hispanic Asian	7.6%	92.4%	7.1%	0.5%	5.3%	1.5%	1.5%	11.6
Other	18.2%	81.8%	14.8%	3.4%	16.1%	1.3%	2.0%	27.0
Socioeconomic Status								
Income								
<$10,000	19.2%	80.8%	15.9%	3.3%	16.5%	1.8%	2.2%	29.3
$10,000-$19,999	18.8%	81.2%	16.0%	2.7%	16.6%	1.7%	1.5%	28.8
$20,000-$29,999	18.8%	81.2%	15.9%	2.9%	16.8%	1.6%	1.4%	28.7
$30,000-$39,999	19.3%	80.7%	16.6%	2.7%	17.3%	1.3%	1.4%	29.4
$40,000-$49,999	18.9%	81.1%	15.9%	3.0%	17.1%	1.1%	1.4%	29.2
$50,000+	15.3%	84.7%	13.3%	2.0%	14.2%	0.8%	1.0%	24.5
Education								
<12 years	17.5%	82.5%	14.5%	3.0%	15.1%	1.8%	1.8%	27.6
12 years	19.7%	80.3%	17.0%	2.8%	17.8%	1.4%	1.5%	30.4
13-15 years	19.8%	80.2%	16.8%	2.9%	17.8%	1.3%	1.5%	29.9
16 years	14.2%	85.8%	12.1%	2.0%	13.1%	0.8%	0.9%	22.1
17+ years	13.9%	86.1%	12.1%	1.8%	12.6%	0.8%	1.0%	21.5
Health Behaviors								
Current drinking status								
Abstainer	11.1%	88.9%	9.6%	1.5%	9.4%	1.3%	1.3%	16.9
Lifetime infrequent	15.6%	84.4%	13.9%	1.7%	13.6%	1.3%	1.2%	24.7
Former	22.3%	77.7%	18.6%	3.6%	20.0%	1.7%	1.2%	36.4
Current (<1 drink/day)	19.1%	80.9%	16.2%	2.9%	17.3%	1.2%	1.4%	28.9
Current (1-<2 drink/day)	19.0%	81.0%	16.1%	3.0%	17.3%	1.5%	1.3%	28.0
Current (2 - <3 drink/day)	23.7%	76.3%	19.4%	4.3%	21.8%	0.8%	1.7%	32.2
Current (3+drink/day)	26.8%	73.2%	21.7%	5.2%	24.5%	1.6%	2.3%	41.7
Smoking status								
Never	14.7%	85.3%	12.9%	1.9%	13.0%	1.1%	1.2%	22.6
Former	17.6%	82.4%	15.0%	2.6%	15.9%	1.2%	1.3%	28.2
Current	23.9%	76.1%	19.7%	4.1%	21.4%	1.8%	2.0%	36.2
Psychosocial factors								
Marital status								
Married	18.7%	81.3%	15.9%	2.8%	16.8%	1.4%	1.5%	28.7
Widowed	9.3%	90.7%	8.4%	0.9%	7.9%	0.8%	1.0%	15.8
Divorced	22.2%	77.7%	18.4%	3.9%	20.5%	1.4%	1.7%	34.6
Never married	18.3%	81.7%	15.7%	2.6%	16.1%	1.7%	1.6%	28.0
Ever married to problem drinker	31.9%	68.1%	25.6%	6.3%	28.5%	2.4%	3.4%	49.5
Current living arrangement								
Live with drinker	19.5%	80.5%	16.7%	2.9%	17.5%	1.3%	1.6%	29.8
Live with heavy drinker	38.0%	62.0%	30.6%	7.4%	34.6%	2.7%	3.4%	64.0
Moderate/heavy drinker	25.2%	74.8%	20.7%	4.5%	22.8%	1.8%	2.1%	39.6
Former drinker	22.9%	77.1%	19.0%	3.9%	20.3%	1.5%	2.5%	37.5

Source: 1988 NHIS Alcohol Supplement

Notes: Percentages adjust for complex sampling design. N=41, 308.

TABLE 2. Mortality Risk (Hazard Ratios) Associated with Childhood Exposure to Problem Drinking, U.S. Adults Ages 21 and Above, 1988-2006

	Model 1	Model 2	Model 3	Model 4
Lived with problem drinker during first 18 years of life	**1.18 *****	**1.16 *****	**1.10 ****	**1.09 ****
Sociodemographic Factors				
Male	1.56 ***	1.67 ***	1.50 ***	1.55 ***
Race (non-Hispanic white)				
Non-Hispanic black	1.24 ***	1.05	1.04	1.03
Hispanic	0.90 +	0.79 ***	0.81 ***	0.81 ***
Non-Hispanic Asian	0.56 ***	0.54 ***	0.58 **	0.58 **
Other	1.08 *	1.03	1.02	1.01
Socioeconomic Status				
Education		0.98 ***	0.99 **	0.99 **
Income		0.79 ***	0.81 ***	0.83 ***
Health Behaviors				
Drinking status (current, <1 per day)				
Abstainer			1.13 ***	1.15 ***
Infrequent			1.13 **	1.13 ***
Former			1.24 ***	1.24 ***
Current, 1-<2 per day			1.10 +	1.10 +
Current, 2-<3 per day			1.48 ***	1.47 ***
Current, 3+ per day			1.57 ***	1.55 ***
Smoking status (never)				
Former			1.24 ***	1.23 ***
Current			2.07 ***	2.04 ***
Psychosocial factors				
Marital status (married)				
Widowed				1.07 *
Divorced/separated				1.14 ***
Single				1.16 ***
Ever married or lived with problem drinker				1.16 ***

Source: 1988-2006 NHIS-LMF.

Notes: Referent in parentheses. Models adjust for complex sampling design. N=41,308 (with 10,494 deaths).

+ p < .10; * p < .05; ** p < .01; *** p < .001.

TABLE 3. Mortality Risk (Hazard Ratios) Associated with Childhood Exposure to Problem Drinking, U.S. Adults Ages 21 and Above, 1988-2006

	Model 1	Model 2	Model 3	Model 4	Model 1	Model 2	Model 3	Model 4	Model 1	Model 2	Model 3	Model 4
	Panel A: Ages 21-44 (N=21,950; 1,030 deaths)				Panel B: Ages 45-64 (N=10,549; 2,812 deaths)				Panel C: Ages 65 and over (N=8,809; 6,652 deaths)			
Lived with problem drinker during first 18 years of life	1.36 ***	1.25 **	1.16 +	1.14 +	1.23 ***	1.20 **	1.12 *	1.11 *	1.10 +	1.09 +	1.06	1.05
Sociodemographic Factors												
Male	1.97 ***	2.16 ***	1.94 ***	2.06 ***	1.46 ***	1.61 ***	1.42 ***	1.46 ***	1.56 ***	1.62 ***	1.48 ***	1.53 ***
Race (non-Hispanic white)												
Non-Hispanic black	2.19 ***	1.67 ***	1.68 ***	1.61 ***	1.49 ***	1.10 +	1.11 *	1.10 +	0.97	0.88 *	0.87 *	0.86 **
Hispanic	1.27 *	0.89	1.05	1.06	0.83 *	0.65 ***	0.72 **	0.71 **	0.86 +	0.80 **	0.79 **	0.79 **
Non-Hispanic Asian	1.96 +	0.49 *	0.54 +	0.54 +	0.66	0.61	0.72	0.73	0.53 ***	0.52 ***	0.53 ***	0.54 ***
Other	1.21	1.08	1.10	1.10	1.20 *	1.06	1.08	1.07	1.03	1.01	0.98	0.98
Socioeconomic Status												
Education		0.92 ***	0.95 ***	0.94 ***		0.96 ***	0.97 ***	0.96 ***		0.99	1.00	1.00
Income		0.69 ***	0.72 ***	0.79 ***		0.72 ***	0.75 ***	0.77 ***		0.88 ***	0.89 ***	0.90 ***
Health Behaviors												
Drinking status (current, <1 per day)												
Abstainer			1.08	1.15			1.14 +	1.15 +			1.13 **	1.14 **
Infrequent			1.11	1.18			1.11	1.12			1.11 *	1.11 *
Former			1.20 *	1.23 *			1.28 ***	1.28 ***			1.21 ***	1.20 ***
Current, 1-<2 per day			1.10	1.05			1.20 *	1.18 +			1.01	1.01
Current, 2-<3 per day			1.44 *	1.40 +			1.62 ***	1.59 ***			1.32 **	1.30 *
Current, 3+ per day			1.69 ***	1.66 ***			1.73 ***	1.72 ***			1.27 +	1.25
Smoking status (never)												
Former			0.86	0.87			1.38 ***	1.38 ***			1.24 ***	1.23 ***
Current			2.06 ***	2.03 ***			2.37 ***	2.36 ***			1.81 ***	1.78 ***
Psychosocial factors												
Marital status (married)												
Widowed				1.90 *				1.10				1.04
Divorced/separated				1.37 ***				1.07				1.14 *
Single				1.61 ***				1.21 *				0.97
Ever married or lived with problem drinker				1.31 **				1.12 +				1.15 **

Source: 1988-2006 NHIS-LMF

Note: referent in parentheses.

+ p < .10; * p < .05; ** p < .01; *** p < .001.

TABLE 4. Mortality Risk (Hazard Ratios) Associated with Types of Exposure, U.S. Adults Ages 21 and Above, 1988-2006

Problem Drinkers during First 18 Years of Life	Model 1	Model 2	Model 3	Model 4
Number of problem drinkers (0)				
1	1.17 ***	1.16 ***	1.11 **	1.10 **
2+	1.24 *	1.21 +	1.07	1.05
Relationship to problem drinker				
Parent	1.18 ***	1.16 ***	1.10 **	1.09 *
Sibling	1.12	1.09	1.04	1.03
Other relative	1.21 *	1.16 +	1.07	1.06
Number of years lived with problem drinker(s) (0)				
1-11	1.11	1.12 +	1.05	1.04
12+	1.21 ***	1.19 ***	1.13 **	1.12 **

Source: 1988-2006 NHIS-LMF.

Notes: Referent in parentheses. Models adjust for complex sampling design. Each set of variables entered separately. Models sequentially add in the following controls: (1) sex and race ; (2) education and income; (3) drinking status and smoking; (4) marital status and ever married to or lived with problem drinker. N=41,830 (with 10,494 deaths).

+ $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$.

TABLE 5. Mortality Risk (Hazard Ratios) Associated with Childhood Exposure to Problem Drinking by Cause of Death, U.S. Adults Ages 21 and Above, 1988-2006

	Model 1	Model 2	Model 3	Model 4
Effect of living with problem drinker during first 18 years on death due to:				
Heart disease	1.15 *	1.13 *	1.10 +	1.10 +
Cancer	1.22 **	1.20 **	1.12 +	1.10
Cancer excluding lung	1.15 +	1.14 +	1.10	1.09
Chronic lower respiratory diseases and lung cancer	1.39 ***	1.35 ***	1.16 +	1.15
External causes	1.53 **	1.45 **	1.33 *	1.31 *
Transport accident	1.73 *	1.64 *	1.64 *	1.61 *
Nontransport accident	1.55 +	1.47	1.35	1.33
Suicide	1.41	1.36	1.16	1.09
Homicide	0.88	0.83	0.67	0.73
Other causes	1.09	1.09	1.04	1.03

Source: 1988-2006 NHIS-LMF.
Notes: Models sequentially add in the following controls: (1) sex and race; (2) education and income; (3) drinking status and smoking; (4) marital status and ever married to or lived with problem drinker. Models adjust for complex sampling design. N=41,308 (with 3,465 heart 2,453 cancer 536 resipiratory, and 396 external deaths, and 3,644 deaths of other causes).
+ $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$.